# CURRENT LITERATURE

## SLEEP AND EPILEPSY

### Distribution of Partial Seizures During the Sleep–Wake Cycle: Differences by Seizure Onset Site

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OBJECTIVE: To evaluate the effects of sleep on partial seizures arising from various brain regions.

METHODS: The authors prospectively studied 133 patients with localization-related epilepsy undergoing video-electroencephalogram (EEG) monitoring over a two-year period. Seizure type, site of onset, sleep/wake state at onset, duration, and epilepsy syndrome diagnosis were recorded. Periorbital, chin electromyogram (EMG), and scalp/sphenoidal electrodes were used. A subset of 34 patients underwent all-night polysomnography with scoring of sleep stages.

RESULTS: The authors analyzed 613 seizures in 133 patients. Forty-three percent (264 of 613) of all partial seizures began during sleep. Sleep seizures began during stages one (23%) and two (68%) but were rare in slow-wave sleep; no seizures occurred during rapid eye movement (REM) sleep. Temporal lobe complex partial seizures were more likely to secondarily generalize during sleep (31%) than during wakefulness (15%), but frontal lobe seizures were less likely to secondarily generalize during sleep (10% versus 26%; $p < 0.005$).

CONCLUSIONS: Partial-onset seizures occur frequently during non-REM (NREM) sleep, especially stage two sleep. Frontal lobe seizures are most likely to occur during sleep. Patients with temporal lobe seizures have intermediate sleep seizure rates, and patients with seizures arising from the occipital or parietal lobes have rare sleep-onset seizures. Sleep, particularly stage 2 sleep, promotes secondary generalization of temporal and occipitoparietal, but not frontal, seizures. These findings suggest that the hypersynchrony of sleep facilitates both initiation and propagation of partial seizures, and that effects of sleep depend in part on the location of the epileptic focus.

### COMMENTARY

To understand the interactions between sleep and epilepsy, it is important to view the timing of seizures during the circadian sleep-wake cycle, that is, whether the seizures tend to occur during the day (diurnal), during the night (nocturnal) or during the day and the night (random). The study of Janz (1), later reproduced by Billiard (2), were the first comprehensive efforts to look at the distribution of generalized seizures during the 24 hour sleep-wake period and were termed "sleep," "day," or "diffuse" epilepsies.

Studies to date, including those of Bazil (2), Crespel et al. (3) and Sammaritano (4), showed that the majority of nocturnal partial seizures occur predominantly during stage two of slow wave sleep and do not occur during rapid eye movement (REM) sleep. Frontal lobe seizures (FLE) are more likely to occur during nighttime sleep, whereas temporal lobe seizures (TLE), although less likely to occur during the night, when they do occur, are more likely to secondarily generalize (3,5).

A modification of sleep architecture is found in patients with TLE and ETL epilepsy both during nights without seizures and more severely in nights with seizures (6,7,8). In fact several authors have proposed that there may be sleep disorder intrinsic to the temporal lobe epilepsy itself (9,10). However, the sleep architecture disruption found in the TLE patients is more severe when compared with that of ETE group (3,6,7). Could the more severe sleep disruption help to promote the secondary generalization that occurs more often in the TLE group than the ETE group? Would the increase in spindle production or synchronization occurring prior to the onset of a seizure as recorded by polysomnography with temporal lobe epilepsy patients also play a role (4)?

This article makes a major contribution to the study of the circadian rhythm of partial seizures. The study is comprehensive: large number of patients; video-EEG long-term monitoring including a subset of these same patients with TLE studied with polysomnography; more precise division of TLE patients into mesial temporal lobe and neocortical temporal lobe epilepsy. The unique finding not tested previously is that while sleep facilitates the initiation and propagation of seizures, there is a partial dependence on the location of the epileptogenic zone, and that changes in neuronal synchrony during sleep are different in each cortical region. Although the

mechanisms for these state dependent changes are not known, future studies will hopefully be rewarding.

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